**Histoplasma capsulatum**

Case

1. 5 works presented with a 1-week history of fever, chills night sweats, cough, headache, fatigue, myalgia and weight loss
2. 2 weeks before onset of symptoms, these 5 workers had begun partial demolition of an abadoned building in a small city near the Ohio River. There was a lot of pigeon dropping around the building. None of the workers wore protective equiptment

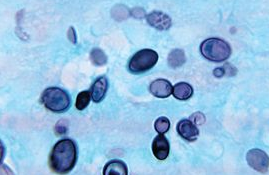
Physical exam

1. CXR: enlarged hilar and mediastinal nodes and multiple nodular infiltrates

DDx

1. Blastomycosis
2. Coccidiomycosis
3. Cryptococcosis
4. Histoplasmosis
5. Legionellosis and other atypical pneumonia
6. Nocardiosis
7. Pneumococcal pneumonia
8. Tuberculosis
9. Rationale: epidemoiological history is classic for histoplasmosis (pigeon dropping). Blasto and crypto are less common and coccidio is not found in that region.

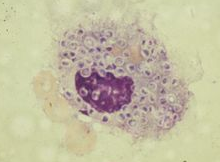
Micro Properties

1. Histoplasma capsulatum is a dimorphic fungus—mold form in soil and yeast in human host
2. Yeast are seen in tissues as thin-walled oval structures visualized by staining with Giemsa or Wright stains of smears of bone marrow, sputum, blood, or lung biopsy
   1. 
3. Mold can be cultured on mycologic agar may take up to 4 weeks of incubation.
   1. The aerial mycelial growth produces characteristic macroconidia (thick wall; finger-like projections) and microconidia.
   2. The mold produces two glycoproteins, H and M, detectable by the agar immunodiffusion test.
   3. Fungal antigens can be detected in serum or urine. The test is very specific, and often results are available much sooner than culture results.

Epidemiology

1. Endemic in parts of central and eason US along Ohio and Mississippi river valleys. –also small populations found in Central and South America, Africa, India, and SE Asia.
2. Grows in soil contaminated with bat or bird droppings—esp pigeon, blackbird, and starling
3. Conidia become airborne when contaminated soil is disturbed and infection results from inhalation
4. High risk groups
   1. Immunologically naïve individuals going to endemic areas
   2. Immuncocompromised persons (cancer, transplant, AIDS)

Pathogenesis

1. The respiratory tract is the portal of entry for H. capsulatum in most patients.
2. Small microconidia reach the alveoli🡪they bind to the CD2/CD18 family of integrins 🡪 engulfed by both PMN’s and macrophages🡪 microconidia transform into budding yeast forms🡪 yeasts grow intracellularly in the inactivated alveolar macrophages 🡪 With time, a granulomatous reaction occurs and produces calcified fibrinous granulomas w/ areas of caseous necrosis in lungs 🡪 yeasts migrate to local draining lymph nodes and then, to distant organs rich in mononuclear phagocytes, such as the reticuloendothelial system, including liver, spleen, and bone marrow
   * 1. -- Histiocyte containing numerous yeast cells of Histoplasma capsulatum.
        (Wright Stain)
   1. The transition from the mycelial to the yeast phase is one of the most critical determinants for establishing infection
   2. The granulomas consist of a mixture of mononuclear phagocytes and lymphocytes, principally T cells.
   3. Cytokine response leads to symptoms such as fatigue and weight loss.

Treatment

1. Most are **self-limiting** and do not require therapy.
2. Oral **itraconazole** for less severe manifestations in immunocompetent individuals.
3. Some patients with preexisting lung diseases such as emphysema may fail to recover—even with therapy—developing a chronic lung disease that resembles tuberculosis. Permanent lung damage may occur.
4. In immunocompromised patients (e.g., those with AIDS), the initial pulmonary infection may disseminate, producing extrapulmonary manifestations that affect mucosal surfaces, liver, spleen, adrenal gland, and meninges. For these **patients with severe symptoms and disseminated histoplasmosis, intravenous amphotericin B** is the drug of choice.

Prevention

1. There is no vaccine available
2. Risk can be lessened in highly contaminated environments by use of personal protective gear.